Letter to the Editor

Treatment with propranolol of 6 patients with idiopathic aquagenic pruritus

To the Editor:

Idiopathic aquagenic pruritus (IAP), as defined by Steinman and Greaves[1] in 1985, occurs after contact with water, involving intense itching without visible skin changes and without an underlying pathology (polycythemia vera, Hodgkin disease, and blood disorders) or drugs that could induce this symptom. Conventional treatments are the addition of sodium bicarbonate to bath water, antihistamines, or phototherapy, which relieve symptoms in 24%, 47%, and 50% of patients, respectively.[2,3] However, propranolol considerably reduced symptoms in 2 patients.[4] Our study, which was conducted in 2 French allergology departments, evaluated the efficacy and tolerance of a 3-month treatment with propranolol in 6 patients with IAP (according to the criteria of Steinman and Greaves[1]). Treatment efficacy was assessed by the percentage improvement in symptoms rated by each patient using a visual analog scale at 1, 4, and 12 weeks of treatment.

Three men and 3 women were included. The average age was 41 years (range, 15-60 years), and the average duration of symptoms at initiation of propranolol was 18 years (10-30 years). Before the introduction of β-blockers, the treatments prescribed were alkalinization of the bath water (never used by patients), emollients (3 cases, ineffective), antihistamines (6 cases, 50% efficiency for 1 case, relapse after stopping), narrow-band UVB therapy (1 case, 100% efficiency, relapse after stopping), fluoxetine (2 cases, ineffective), and clonidine (1 case, ineffective).

The patients received 10 to 40 mg/d propranolol for 3 months, depending on their tolerance (Table I). In less than 7 days, complete remission was obtained in 4 patients, and symptoms decreased by 90% for 1 patient. No relapse occurred within 3 months of treatment, but after discontinuation of propranolol, clinical signs recurred in 5 patients. A cough was the only reported side effect induced by voluntarily doubling the dose of β-blocker (from 20 to 40 mg/d). The patient who did not respond to propranolol is also the one for whom clonidine was ineffective. On relapse, 1 patient was retreated with propranolol and experienced the same improvement seen with the first course of propranolol.

The pathophysiology of AIP is unknown, but inappropriate activation of the autonomic nervous system could be involved. Neurotransmitters, such as acetylcholine and vasoactive intestinal peptide, are indeed released in the skin on contact with water in patients with IAP, whereas the role of histamine appears to be minimal; antihistamines have a low efficiency as well.[5,6]

According to our results (improvement of >90% in 5/6 patients with minimal side effects), the β-blocker appears more effective and better accepted than conventional treatments. The therapeutic effect of propranolol, a β-receptor antagonist of adrenaline, suggests involvement of the sympathetic system in the occurrence of IAP. This anomaly does not explain all cases of IAP because 1 patient responded to neither propranolol nor clonidine (α_2-adrenergic agonist), the effect of which is to decrease the sympathetic tone.

TABLE I. Six patients treated with propranolol for 3 months

Patient/ year of birth/ sex	Dose of propranolol (mg)	Improvement with treatment (%)	Side effects	Relapse after stopping propranolol	Former clonidine treatment
1/1946/F	10	90	No	Yes	No
2/1972/M	20	0	No	No	Yes
3/1978/M	20	100	No	Yes	No
4/1995/M	40	100	No	Yes	No
5/1951/F	40	100	Cough	Yes	No
6/1961/F	40	100	No	Yes	No

F, Female; *M*, male.

Propranolol at a dosage of 10 to 40 mg/d could be useful in the treatment of IAP presenting a typical clinical picture and in the absence of contraindications to β-blockers. Its prescription should not be delayed because of the lack of efficiency of conventional treatments for this disease.

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